a history of massive hemorrhage occurring on several occasions over a long period of time with no progression to cachexia or appearance of other diagnostic symptoms and signs. In most cases a filling defect can be observed in roentgen studies, and the benign nature of the tumor is suggested by the lack of induration of the wall of the stomach adjacent to the base of the tumor and by the fact that there is normal motility in the stomach wall up to the edge of the tumor-involved area.

The radiological findings in the case of schwannoma of the stomach observed by the authors and reported herein were similar, if not identical, to those associated with other benign tumors. There is no true diagnostic or differentiating point. The benign tumors, in general, appear as a well-defined sessile or pedunculated, rounded mass projecting into the lumen of the stomach. They may be as small as a small polyp or papilloma or so large as to fill the entire gastric lumen. Usually there is no disturbance in gastric motility and no invasion of the stomach wall. However, at times, the size and position of the tumor may be such as to cause partial pyloric obstruction. In the case of the larger tumors, an ulcer crater is frequently present at the apex of the mass. Perier, in a report of three cases of schwannoma of the stomach, described roentgenologic findings the same as those noted in the case reported herein, but added that similar findings may also be present in connection with leiomyomas. It is impossible to determine precisely the kind of lesion on the basis of roentgen evidence.

As benign tumors of the stomach may undergo malignant degeneration which is not at first detectable radiologically, operative intervention may not safely be delayed. The treatment, once the distressing symptoms are controlled, is excision. This may be a localized procedure, or, if there is question of malignant change, more radical stomach resection.

CASE REPORT

A 61-year-old white male entered the hospital Dec. 4, 1949, with complaint of tarry stools and weakness and fainting for three days. The body weight of the patient had decreased by 35 pounds in the preceding five years, but anorexia was not noted. Except for pallor, no abnormalities were observed in physical examination. The blood pressure was 100 mm. of mercury systolic and 50 mm. diastolic and the pulse rate was 120 per minute. The patient was treated supportively and several blood transfusions were given. When sufficient recovery had occurred, roentgen studies of the stomach were carried out. A large, intraluminal mass arising from the mid-portion of the lesser curvature was observed. The radiologic diagnosis was leiomyoma with ulceration of the tumor mass. Diverticulum of the duodenum was also noted. Results of gastric analysis were within normal limits of acidity. Seventeen days after the patient was admitted, the growth was removed en bloc by subtotal resection.

The resected segment of stomach was annular, 16 cm. at the greatest diameter. The stomach wall contained a submucosal and circumscribed, gray, rather soft nodule 3 cm. in diameter. A covering of ulcerated mucosa was observed in microscopic examination of a section of the tumor mass which lay in the mid-portion of the greater curvature of the stomach on the anterior side. The ulcer was covered with granulation tissue which rested on the large nodule in the stomach wall. The mucosa at the ulcer margin was of normal appearance. The tumor mass was made up of elongated cellular fibers with pronounced palisading and regimentation of the nuclei. The nuclei were elongated with tapering ends, had vesicular chromatin patterns, and approximated each other in size. There was a clear zone of cytoplasm at both poles of the cells. In the space between the regimented nuclei there was a loose arrangement of connective tissue cells with round, oval and elongated nuclei. The tissue was cellular throughout, but no malignant changes were observed. The pathologic diagnosis was neurilemmoma (schwannoma of the stomach), with chronic ulcer over the neurilemmoma.

The patient recovered and was discharbed on the eighth postoperative day.

SUMMARY

A case of schwannoma of the stomach with overlying gastric ulcers is reported. The tumor was removed by subtotal resection and the patient recovered.

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REFERENCES

Askanazy: Cited by Willius.

Kiefer, E. D.: Benign tumors of the stomach, S. Clin. N. Amer., 21:711, June 1941.

Laird, W. R., and Nolan, L. E.: Significance of schwannomas, Am. J. of Surg., 61:418-420, Sept. 1943.

Masson, P., Experimental and spontaneous schwannomas, Am. J. Path., 8:367-389, July 1932.

Perier, E. A.: Schwannoma of the stomach, Presse Med., 53:134-135, March 17, 1945.

Stout, A. P.: The peripheral manifestations of the specific nerve sheath tumor (neurilemmoma), Am. J. Cancer, 24:751, Aug. 1935.

Verocay: Cited by Willius.

West, J. P., and Knox, G.: Neurogenic tumors of the stomach, Surgery, 23:450, March 1948.

Willius, R. A.: Pathology of Tumours, pp. 828-835, Butterworth & Co., Ltd., London, 1948.

Skin Granuloma Following Laceration by a Fluorescent Lamp

Report of a Case

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SKIN lesions associated with exposure to beryllium compounds have been described mainly in conjunction with reports of pulmonary lesions found in industrial workers exposed to these compounds.[5, 12] Recent descriptions of subcutaneous granulomas at the sites of skin lacerations with fluorescent lamp fragments carry the problem, in some measure, from the industrial plant into the office and home. Seven such cases have been reported.[1, 6, 9, 10] Although the use of beryllium silicate, the offending substance, is presumably being discontinued by most manufacturers, the lamps in current use and in stock will present a danger for some time to come.[13]

CASE REPORT

In September 1948 a white 7-year-old boy received a single laceration approximately 1 cm. in length over the dorsal aspect of the proximal interphalangeal joint of the left middle finger from a broken fluorescent lamp. Treatment consisted of mercurochrome, applied at the time of injury; boric soaks, advised one week later when mild cellulitis was observed; and, when a moderate painless swelling occurred, a five-day period of penicillin therapy, warm compresses and bed rest. In spite of active treatment the lacerated area became progressively indurated, and by December 1948 a thickened, reddish, slightly tender granulomatous lesion had developed which intermittently exuded small amounts of thin, clear fluid (Figure 1).

Intradermal tuberculin and coccidioidin tests (each 1:1000) elicited negative reactions. The blood serum gave a negative

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reaction to the Kolmer test and to agglutination tests for typhoid, paratyphoid A and B, tularemia and brucellosis. In a radiologic examination no evidence of foreign body or bone destruction in the finger was noted; there was some suggestion of effusion in the proximal interphalangeal joint space. A roentgen film of the chest was normal.

In a biopsy specimen from the lesion in February 1949 portions of tuberculoid granulomas infiltrated with chronic inflammatory cells were observed. A few foreign body giant cells were noted, one of which contained a small triangular crystalline body. Nonhemolytic Staphylococcus albus and diphtheroid bacilli were grown on cultures of the biopsy material. In tests for acid-fast organisms, including guinea pig inoculation, tubercle bacilli were not demonstrated. In March 1949 the patient received a series of x-ray treatments to the involved finger (450 r in air).

When the patient was admitted to the University of California Hospital in April 1949 the fibrous granuloma measured 2 by 3 cm. and there was a 0.5 cm. patch of ulceration in the central portion of it. The area was not particularly tender and there was full range of motion of the proximal interphalangeal joint.

Spectrographic studies of the urine for beryllium content were not done. Radiologic studies of the finger again showed no evidence of foreign body, bone destruction or joint involvement. The soft tissue swelling was noted to be somewhat larger than on the previous examination.

The lesion was excised, including a 1 cm. margin beyond its indurated borders. The dissection was carried down to the extensor tendon, where it was apparent that the joint capsule was elevated and somewhat tense. The capsule was incised, allowing the escape of a small amount of clear fluid. A small mass of tissue, grossly resembling that of the choroid plexus, extended into the joint space and was excised. After closure of the capsule, the resultant defect on the dorsum of the finger was covered by a full thickness skin graft procured from the lateral anterior abdominal wall.

In microscopic study of the specimen (Figure 2) numbers of small tuberculoid granulomas were noted, each consisting of clusters of epithelioid cells lying in a fibrous stroma. In many of the tubercles there were central areas of hyalin-like necrosis. No giant cells or acid-fast organisms were noted. No glass particles were discovered in the specimen. A pathologic diagnosis of chronic granuloma of the finger, compatible with beryllium granuloma, was made.

Specimens of tissue and fluid removed from within the joint were tested for bacterial content by cultures and guinea pig inoculation but no organisms were demonstrated.

A portion of the specimen was examined spectrographically by a method described by Cholak and Hubbard.[2] No beryllium was found. (It should be noted that Cholak and Hubbard indicated that the lower limit of detectability by this method is 0.25 micrograms.) It was assumed that a smaller, undetermined amount of beryllium was present in the excised lesion.

The postoperative course was uneventful. The skin graft has healed satisfactorily. Although the joint area remained somewhat enlarged, there was normal, painless range of motion (Figure 3). In later x-ray studies of the finger no evidence of bone erosion or destruction was observed.

SUMMARY

A skin granuloma occurred at the site of laceration of a finger with a fluorescent lamp fragment. Because of certain characteristics noted in specimens examined microscopically, beryllium granuloma was diagnosed (although it could not be confirmed spectrographically) when results of tests to demonstrate other etiologic factors were negative. The treatment was wide excision of the lesion. In subsequent x-ray studies there was no evidence of involvement of the bone.

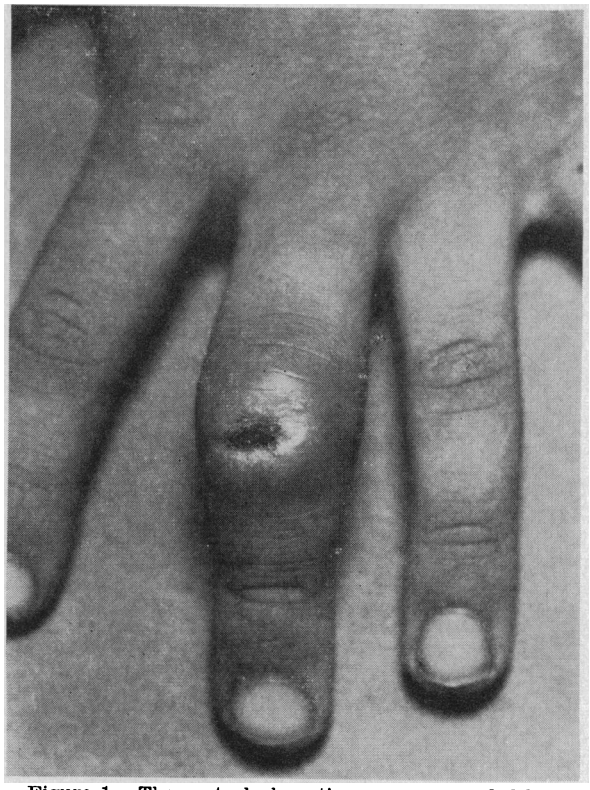

Figure 1.—The central ulceration was surrounded by an indurated, swollen, chronically inflamed area.

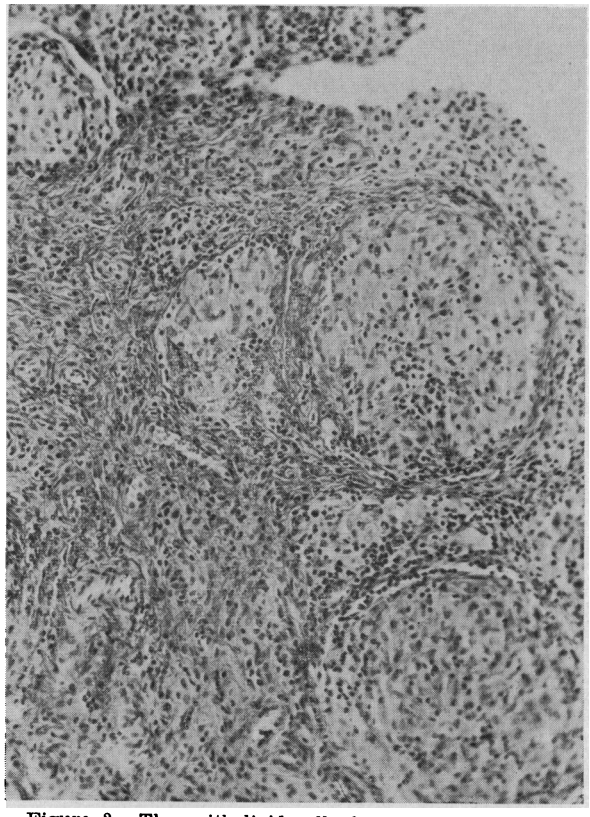

Figure 2.—The epithelioid cells form small granulomas which are strikingly similar to those found in tuberculous lesions.

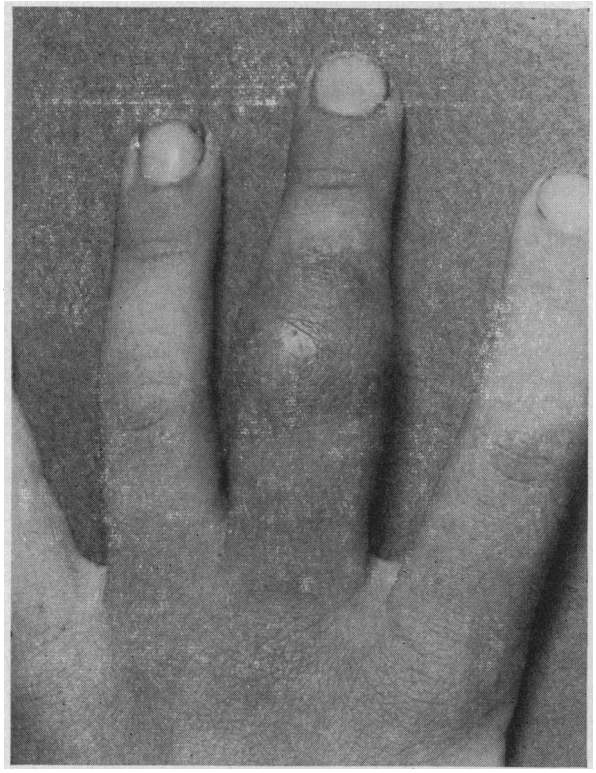

Figure 3.—The postoperative photograph illustrates the size of the skin graft necessary to close the wound. Wide surgical excision of the beryllium granuloma is mandatory.

REFERENCES

1. Coakley, W. A., Shapiro, R., and Robertson, G. W.: Granuloma of the skin at site of injury by a fluorescent bulb, J.A.M.A., 139:1147-1148, April 23, 1949.

2. Cholak, J., and Hubbard, D. M.: Spectrographic determination of beryllium in biological material and in air, Analytical Chemistry, 20:73, Jan. 1948.

3. Fairhall, L. T., et al.: The Toxicology of Beryllium, Bulletin 181, National Institute of Health, Division of Industrial Hygiene, 1943.

4. Forsythe, W. E., and Adams, E. Q.: Fluorescent and Other Gaseous Discharge Lamps, New York and Toronto, Murray Hill Books, Inc., 1948.

5. Gilman, I.: Beryllium (Glucinium). Occupation and Health Supplement, International Labor Office, Geneva, 1938.

6. Grier, R. S., Nash, P., and Freiman, D. G.: Skin lesions in persons exposed to beryllium compounds, J. Indust. Hyg. and Toxicol., 30:228-237, July 1948.

7. Hardy, H. L., and Tabershaw, I. R.: Delayed chemical pneumonitis occurring in workers exposed to beryllium compounds, J. Indust. Hyg. and Toxicol., 28:197-211, Sept. 1946.

8. Martland, H. S., Brodkin, H. A., and Martland, H. S., Jr.: Occupational beryllium poisoning in New Jersey, J. M. Soc. New Jersey, 45:5-14, Jan. 1948.

9. Nichol, A. D., and Dominguez, R.: Cutaneous granuloma from accidental contamination with beryllium phosphors, J.A.M.A., 140:855, July 9, 1949.

10. Shook, C. F., and Powell, J. P.: A beryllium case, Indust. Med., 17:403, Oct. 1948.

11. Titus, A. C.: Air contamination during machining of beryllium stainless steel. J. Indust. Hyg. and Toxicol., 30:29-31, Jan. 1948.

12. Van Ordstrand, H. S., Hughes, R., De Nardi, J. M., and Carmody, M. G.: Beryllium poisoning, J.A.M.A., 129:1084-1090, Dec. 15, 1945.

13. Wounds contaminated by beryllium phosphors, Queries and Minor Notes, J.A.M.A., 139:1047, April 9, 1949.

Treatment of Diabetes Insipidus with Propylthiouracil

Report of a Case

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THE work of Mahoney and Sheehan[1] showing the abolition of experimentally produced polyuria and polydipsia in dogs by total thyroidectomy and the reestablishment of the symptoms following orally administered desiccated whole thyroid gland, suggested the use of a thyroid-inhibiting drug, propylthiouracil, in the treatment of this syndrome.

CASE REPORT

A white woman, 48 years old, who for two years had had severe headaches frequently accompanied by nausea and vomiting, had been admitted several times to the hospital with a diagnosis of migraine. These attacks responded more or less well to the usual therapeutic measures, and the patient was able to leave the hospital after three or four days on each occasion. Quite abruptly insatiable thirst developed and the patient urinated as often as every 15 minutes. Upon physical examination no abnormality was noted. The 24-hour volume of urine was in excess of 4,000 cc. and the specific gravity was very low. Treatment with Pitressin® tannate in oil relieved the polyuria and polydipsia for from 18 to 36 hours. The 24-hour output fell to less than 1,800 cc., and the specific gravity rose to more normal levels. Each dose of Pitressin® or Pituitrin,® which also was given, was followed by headaches, nausea and vomiting.

Propylthiouracil was given in a dose of 150 mg. daily. After three days the patient had no headache, nausea, vomiting, thirst, or frequency of urination. With no other medication than propylthiouracil the patient was symptom-free for 12 weeks except for two brief periods in which the drug was omitted and polyuria and polydipsia promptly recurred. While taking propylthiouracil the patient urinated only once every five or six hours; the specific gravity of the urine was greater than 1.015, and the 24-hour output was less than 1,800 cc.

Propylthiouracil was discontinued after five months, and at last report six weeks later there had been no recurrence of polyuria or polydipsia.

SUMMARY

Polyuria and polydipsia developed suddenly in a patient who had been hospitalized several times with severe headaches, nausea and vomiting. Temporary remission of the polyuria and thirst was obtained with Pitressin,® but each dose was followed by headache and nausea.

Propylthiouracil, 150 mg. daily, was given, and after three days all symptoms abated. With no other medication the patient was symptom-free for the next 12 weeks save for two brief periods when the drug was omitted and polyuria and polydipsia promptly recurred. The drug was finally discontinued after five months; and the patient, when last observed six weeks later, had had no recurrence of excessive urination or thirst.

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REFERENCE

1. Mahoney, W., and Sheehan, D.: The effect of total thyroidectomy upon experimental diabetes insipidus in dogs, Am. J. Physiol., 112:250, 1935.